Tansy ragwort poisoning in a horse in southern Ontario

Véronique de Lanux-Van Gorder

Abstract — Bizarre behavior, apparent lameness, and colic were noticed in 1 of 3 horses on a pasture overgrown by weeds during a drought. Liver failure and hepatoencephalopathy were diagnosed, caused by pyrrolizidine alkaloid toxicosis associated with consumption of tansy ragwort. The horse made a full recovery when removed from the pasture.

Résumé — Empoisonnment à l'herbe de Saint-Jacques «*Senecio jacobaea*» chez un cheval du sud de l'Ontario. Un comportement bizarre, une boiterie évidente et une colique ont été observés chez un des 3 chevaux d'un pâturage envahi de mauvaises herbes au cours d'une sécheresse. Une défaillance hépatique et de l'hépato-encéphalopathie ont été diagnostiquées suite à une intoxication à la pyrrolizidine, un alcaloïde contenu dans l'herbe Saint-Jacques. Le cheval s'est parfaitement rétabli lorsqu'il a été retiré du pâturage.

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A 26-year-old hunter gelding, 1 of 3 horses on a farm in Wellington County, was found by his owners walking aimlessly and staggering repeatedly through a wooden fence, causing lacerations and contusions to the brisket area. The horse did not seem to recognize his owners, who tried unsuccessfully for 2 h to direct him away from the fences, until he collapsed in lateral recumbency.

When examined that evening, the horse was standing, depressed, and periodically circling to the left, repeatedly flexing the left hind leg and showing evidence of lameness. The abdomen was tucked up and the horse periodically looked at his left flank. A few normal-looking fecal balls were passed during the examination. The rectal temperature was 38.6°C, the heart rate was 36 beats/min, and the respiratory rate was 10 breaths/min. Other observations included dark oral mucous membranes, multiple oral ulcers (2 to 40 mm in diameter), and ptyalism. The horse was treated with mineral oil, 4 L, by stomach tube, and detomidine (Dormosedan, SmithKline Beecham, Philadelphia, Pennsylvania, USA), 1 mL, IV.

The following day (Day 2), the horse appeared depressed, standing with his head low, but was apparently eating and drinking normally, with no evidence of nervous dysfunction or lameness. Bowel sounds were normal. Rectal temperature was 37.6°C, the heart rate was 45 beats/min, and the respiratory rate was 24 breaths/min. The gingival and buccal mucosal ulcers were healing. Blood samples were obtained by jugular venipuncture for a complete blood cell count (CBC) and biochemical profile. Preliminary results showed increases in aspartate aminotransferase (AST) (776 U/L; reference range, 160 to 480 U/L) and total protein (88 g/L; reference range, 56 to 79 g/L). Urea was low (3.12 mmol/L; reference range, 3.57 to 10.7 mmol/L). The CBC showed neutrophilia (19.5×10^9 cells/L; reference range, 2.1 to 6.0×10^9 cells/L) and lymphopenia (0.98×10^9 cell/L; reference range, 1.7 to 5.0×10^9 cells/L).

On Day 3, the horse remained depressed but was apparently eating and drinking normally. The heart rate was 45 beats/min, the respiratory rate was 18 breaths/min, and the rectal temperature was 38.8°C. Additional blood samples were collected. Abnormalities in the biochemical profile included high postprandial serum bile acids (BAs) (39 mmol/L; reference range, 4 to 10 mmol/L), total protein (81 g/L; reference range, 56–79 g/L), globulin (55 g/L; reference range, 21 to 40 g/L), alkaline phosphatase (ALP) (773 U/L; reference range, 90 to 235 U/L), γ-glutamyltransferase (GGT) (331 U/L; reference range 7 to 30 U/L), AST (1063 U/L; reference range 160 to 480 U/L), and creatinine kinase (CK) (2077 U/L; reference range, 150 to 500 U/L). Decreases were noted in phosphorus (0.66 mmol/L; reference range, 0.9 to 1.8), albumin (26 g/L; reference range, 30 to 38 g/L), urea (2.8 mmol/L; 3.57 to 10.7 mmol/L), and haptoglobin (0.05 g/L; reference range, 0.50 to 2.00 g/L).

The white blood cell count was high (14.4×10^9 cells/L; reference range, 2.1 to 6.0×10^9 cells/L), with neutrophilia (12.16×10^9 cells/L; reference range, 2.1 to 6.0×10^9 cells/L), lymphopenia (1.43×10^9 cells/L; reference range, 1.7 to 5.0×10^9 cells/L), and basophils were within normal range (0.14×10^9 cells/L; reference range, 0 to 0.1 cells/L). Mean corpuscular volume (MCV) was 52 fL (reference range, 39 to 49 fL), and mean cellular hemoglobin (MCH) was 19 pg (reference range, 15 to 18 pg).

As plant toxicosis was suspected, the field was examined, and 3 plants that grew in abundance on the rocky pasture were collected for identification. The most abundant was identified as tansy ragwort (*Senecio jacobaea*). The others were celery buttercup (cursed crowfoot, poison buttercup) (*Ranunculus acrus sceleratus*), the most toxic plant of the ranunculus family, and

Ontario Veterinary College, University of Guelph, Guelph, Ontario, N1G 2W1.

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tall buttercup *(R. acris)*, both of which may cause oral lesions (1).

On Day 7, the horse was much brighter and more responsive, was drinking and eating normally, and appeared normal to the owner. Most of the oral lesions had healed.

Tansy ragwort is usually unpalatable. However, in periods of drought, such as the one experienced in the spring of 1998 in southern Ontario, horses may eat weeds that easily outgrow grass during harsh conditions (3). The oral lesions seen in this horse suggested that he had been eating weeds such as *R. acris* and *R. scleratus,* and the age of the lesions indicated that this had been occurring for some time before his illness. It was likely that he had also eaten tansy ragwort, which was by far the most abundant weed in the field. The other 2 horses on the farm may have been unaffected, because they ingested a smaller quantity of the pyrrolizidine alkaloids (PAs) associated with tansy ragwort. However, individual animals may vary in their response or resistance to PAs (2).

Pyrrolizidine alkaloid toxicosis associated with consumption of tansy ragwort can result in major economic losses in the livestock industry. Pyrrolizidine alkaloids are metabolized to highly reactive pyrroles that are antimitotic cytotoxins. Hepatocyte nuclei are most commonly affected, causing irreversible liver damage (2–5).

In horses, tansy ragwort poisoning causes hepatic failure days to months after consumption of the plant. Clinical signs may include weight loss; abdominal pain; constipation or diarrhea; photosensitization; jaundice; and hepatic encephalopathy, exhibited by dullness, yawning, aimless walking, head-pressing, ataxia, and occasional mania (3,5,6). The histopathologic hallmarks of tansy ragwort poisoning are megalocytosis of hepatocytes; bile duct hyperplasia; periportal fibrosis; fibrous occlusion of central and hepatic veins, with extension of fibrosis into the sinusoids; centrilobular hemorrhage; and hepatocellular necrosis (3,6–8).

Tansy ragwort is indigenous to Europe and the British Isles and is commonly identified in some western states, such as California, Oregon, and Washington. It was introduced to the Canadian maritime provinces approximately 150 y ago (6). Seeds are easily disseminated by air, water, or on automobile tires; thus, invasion of uncultivated land can readily occur (3). To our knowledge, this is the first report of tansy ragwort in southern Ontario.

The acute onset of neurological dysfunction without prodromal signs, the aged oral lesions, and the unusual abundance of tansy ragwort on the pasture suggested a diagnosis of PA toxicosis (2,3). The high GGT, ALP, AST, and postprandial serum BA levels and the low urea, which indicate liver damage with biliary involvement (2,3,7,8), gave further support to this diagnosis.

Encephalopathy in PA toxicosis is probably caused by an increase in aromatic amino acids and concurrent decrease in branched chain amino acids, secondary to liver damage. This change in amino acid availability results in neurotransmitter disturbance and the unusual behavior seen in this horse (4,5).

Gamma-glutamyltransferase reliably reflects liver damage and is a good early indicator of PA toxicosis (2,3). However, GGT may decrease to preexposure levels, despite progressive liver lesions (1). Elevated serum ALP is reported in tansy ragwort poisoning in horses, peaking at varying times during the disease and often increasing during the terminal stages of liver failure (2). Aspartate aminotransferase lacks specificity for chronic liver disease, but it may remain elevated during liver failure, after other enzymes have returned to normal; in the absence of nonhepatic diseases, this indicates ongoing hepatic necrosis (8).

When clinical signs of PA toxicosis appear, hepatic damage is irreversible (2,3). Serum GGT, ALP, and BAs should be screened when PA toxicosis is suspected, and the liver should be biopsied if these enzymes are high. Enzyme screening should be repeated monthly or bimonthly to monitor liver damage (2); further biopsies are indicated if serum enzymes increase. Postprandial serum BAs $\geq$ 50 mmol/L are considered incompatible with survival. In the case reported here, and in one other case of PA toxicosis (3), a horse with serum BAs $<$ 50 mmol/L recovered.

Pyrrolizidine alkaloid toxicosis may be difficult to prevent, since animals consuming large quantities of weeds show no signs of poisoning until acute disease occurs. Affected animals should be taken off pasture and fed a diet adequate in carbohydrates, but with limited protein, to help decrease gastrointestinal ammonia production and aromatic amino acid accumulation. Oat or timothy hay may be fed to treat diarrhea and ileus associated with liver failure (7).

Tansy ragwort seeds may be disseminated over long distances, and under the right conditions, the plant can proliferate easily, as shown in this case. Tansy ragwort may be present in areas such as southwestern Ontario, previously considered free of this weed; and where tansy ragwort occurs, PA toxicosis should be suspected in a horse with sudden liver failure and hepatoencephalopathy.

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